Zacks Small-Cap Research
Sponsored – Impartial - Comprehensive

November 9, 2022
David Bautz, PhD
(312) 265-9471
dbautz@zacks.com

scr.zacks.com
10 S. Riverside Plaza, Chicago, IL 60606

Satellos Biosciences Inc.

(OTCQB: MSCLF)

MSCLF: Initiating Coverage of Satellos Biosciences; A Muscle Regeneration Company…

Based on our probability adjusted DCF model that takes into account potential future revenues in DMD, MSCLF is valued at $0.70/share. This model is highly dependent upon continued success of the development candidates and will be adjusted accordingly based on future clinical results.

Current Price (11/09/22)	$0.12
Valuation	**$0.70**

OUTLOOK

We are initiating coverage of Satellos Biosciences Inc. (MSCLF) with a $0.70 valuation. Satellos is a biotechnology company dedicated to developing treatments to aid in muscle regeneration. The company's lead program is the development of a small molecule drug for the treatment of Duchenne muscular dystrophy (DMD), a debilitating and progressively worsening muscle degeneration which typically leads to death in the third decade of life. Satellos has identified multiple signaling pathways that could be modulated to affect asymmetric stem cell division and muscle regeneration in DMD, but that may also be amenable to treating other muscular dystrophies. The company has prioritized a target in the Notch signaling pathway with the goal of identifying lead chemical compounds by the end of 2022.

SUMMARY DATA

52-Week High	**$1.14**
52-Week Low	**$0.02**
One-Year Return (%)	**-89.16**
Beta	**2.08**
Average Daily Volume (sh)	**703**
Shares Outstanding (mil)	**42**
Market Capitalization ($mil)	**$5**
Short Interest Ratio (days)	**1**
Institutional Ownership (%)	**0**
Insider Ownership (%)	**N/A**
Annual Cash Dividend	**$0.00**
Dividend Yield (%)	**0.00**
5-Yr. Historical Growth Rates	
Sales (%)	**N/A**
Earnings Per Share (%)	**N/A**
Dividend (%)	**N/A**
P/E using TTM EPS	**N/A**
P/E using 2022 Estimate	**-0.3**
P/E using 2023 Estimate	**-0.2**

Risk Level	**High**
Type of Stock	**N/A**
Industry	**Med-Biomed/Gene**

ZACKS ESTIMATES

Revenue
(In millions of $)

	Q1 (Mar)	Q2 (Jun)	Q3 (Sep)	Q4 (Dec)	Year (Dec)
2021	0.0 A	0.0 A	0.0 A	0.0 A	0.1 A
2022	0.1 A	0.0 A	0.0 E	0.0 E	0.1 E
2023					0.0 E
2024					0.0 E

Earnings per Share

	Q1 (Mar)	Q2 (Jun)	Q3 (Sep)	Q4 (Dec)	Year (Dec)
2021	-$0.16 A	-$0.14 A	-$0.19 A	-$0.05 A	-$0.51 A
2022	-$0.05 A	-$0.05 A	-$0.05 E	-$0.04 E	-$0.20 E
2023					-$0.13 E
2024					-$0.13 E

© Copyright 2022, Zacks Investment Research. All Rights Reserved.

WHAT'S NEW

Initiating Coverage



Source: Satellos Bioscience, Inc.

We are initiating coverage of Satellos Biosciences Inc. (MSCLF) with a $0.70 valuation. Satellos is a biopharmaceutical company developing treatments for muscle diseases, including muscular dystrophies and muscle loss associated with chronic illness and aging. Satellos' core technology is based on discoveries by its scientific co-founder that implicates impairments in muscle stem cell division as a cause of muscle-related diseases. The company has already identified multiple pathways in muscle stem cells that can be targeted for therapeutic intervention and drug discovery efforts are currently underway. The lead indication for Satellos is Duchenne muscular dystrophy (DMD), a rare deadly genetic disease that has limited treatment options, none of which are curative. Proof-of-concept studies in a DMD mouse model showed that targeting muscle stem cells resulted in enhanced muscle quality, performance, and a return to cell division equilibrium. While DMD is the current focus, the company's technology will likely be amenable to treating multiple other conditions, including orphan muscular dystrophies and other muscle wasting conditions.

A New Approach to Treating Muscle Diseases
Satellos co-founder Dr. Michael Rudnicki has been at the forefront of muscle stem cell research for over 20 years. He has co-authored a number of high-profile publications describing the role of muscle stem cells in muscle regeneration along with their role in aging and disease. Satellos is using the knowledge gained from these studies to develop muscle stem cell therapies for multiple indications.

DMD is a Fatal Disease Where New Treatment Options Are Needed
DMD is an X-linked recessive disorder that afflicts approximately 1 in 5000 boys. The average age of diagnosis is five years of age. The disease results in progressive muscle weakness and loss of muscle mass, with most DMD patients being wheelchair bound by the age of 10. While advancements in treatment over the past 30 years have resulted in an increase in survival, those treatments have harsh side effects and most patients succumb to the disease by 30 years of age.

Targeting the Underlying Pathology of DMD
Dr. Rudnicki's work shows that correcting the imbalance in muscle stem cell regeneration in a mouse model of DMD can increase muscle mass and strength. Satellos has identified several biological pathways that can be targeted for therapeutic intervention to bring about this change and is currently working to bring multiple drug candidates into preclinical testing.

Safety Concerns with Gene Therapy Approaches to Treating DMD
Multiple companies are developing gene therapy treatments for DMD that attempt to insert a functional 'microdystrophin' into the patients' cells to restore the function of the mutated dystrophin protein. Previously, Pfizer had to put a clinical hold on its study of a microdystrophin delivered via an adeno-associated virus (AAV) vector due to a patient death in the trial. This follows reports of other serious adverse events in other gene therapy studies in DMD patients. Satellos is developing small molecule drugs to treat DMD, which we believe will ultimately be a safer approach.

Significant Valuation Expansion Possible
Satellos currently has an approximately $10 million market cap and when compared to peer companies that are developing DMD therapies we believe there is the potential for significant valuation expansion, with peer companies in the IND-enabling stage (where Satellos plans to be in the next 12 months) having market caps 10X Satellos' current valuation.

INVESTMENT THESIS

Satellos Bioscience Inc. (MSCLF) is a Canadian biotechnology company dedicated to developing therapies for the treatment of life-threatening, degenerative muscle disorders based on relevant discoveries by the company's scientific co-founder. Satellos is currently focused on the development of a new class of compounds that could help to restore muscle regeneration in those suffering from Duchenne muscular dystrophy (DMD), the most common genetic disorder diagnosed in childhood, with additional applications possible in other degenerative disorders, including orphan and ultra-orphan muscular dystrophies. A number of therapies have recently been approved for DMD, however none is curative and whether they impact the natural progression of the disease is still being tested. Thus, additional treatment options are still urgently needed.

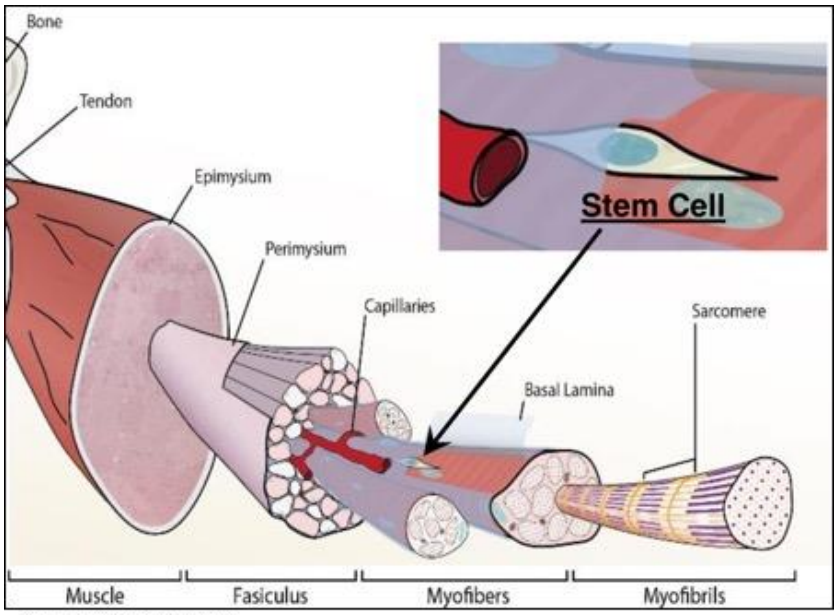
Through studies on muscle stem cells, Satellos' Chief Scientific Officer, Dr. Michael Rudnicki, characterized their role in normal health and how deficiencies in their function can lead to diseases such as DMD. Satellos created a proprietary drug discovery platform, MyoReGenX™, that recapitulates the muscle stem cell environment to aid in identification of regulators of stem cell division. Using this platform, the company has identified multiple signaling pathways that may be amenable to manipulation with drug candidates, with DMD being the initial focus.

DMD results from a lack of dystrophin protein in muscle cells (discussed later in the article), thus many current treatment options are directed toward increasing the amount of dystrophin in a patients' cells. Satellos is taking a different approach that is not predicated on the presence of functional dystrophin. Instead, the company is focused on the role of dystrophin in signal transduction and its role in muscle stem cell polarization and division. Satellos hypothesizes that the lack of dystrophin in DMD results in a decrease in 'asymmetric' cell division, which normally leads to production of muscle progenitor cells, which are required for muscle repair and regeneration. Satellos has identified multiple signaling pathways that could be exploited to correct this issue, and the company has identified multiple compounds that could activate these pathways. Preliminary results with a lead compound targeting a specific pathway showed modulation of this pathway leads to increased muscle strength and performance in a mouse model of DMD. Satellos is the only company we are aware of that is targeting muscle stem cells as a therapeutic approach to DMD, which could ultimately change the entire treatment paradigm for this and other muscle wasting diseases.

Muscle Stem Cells

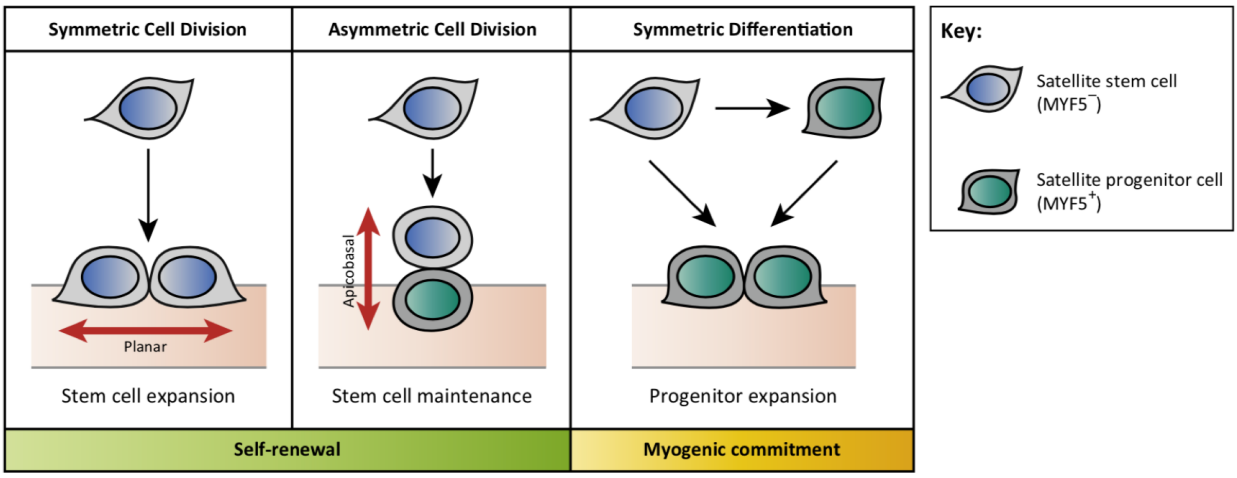
Muscle stem cells (or satellite cells) are required for the normal growth, maintenance, and repair of skeletal muscle. While normally inactive, the cells can be induced to divide following injury. Satellite cells are a heterogeneous population of cells that include both a population of cells capable of self-renewal and a population of progenitor cells capable of muscle repair (Kuang *et al.*, 2007). Satellite cells are located in a niche between the basal lamina and the myofibers. Their polarity leads to differentiated expression of certain molecules on the basal and apical surface of the cell; integrin-A7 and -B1, and dystroglycan are expressed on the basal surface (Blanco-Bose *et al.*, 2001) while M-cadherin and NCAM are expressed on the apical surface (Chargé *et al*., 2004).

© Copyright 2022, Zacks Investment Research. All Rights Reserved.



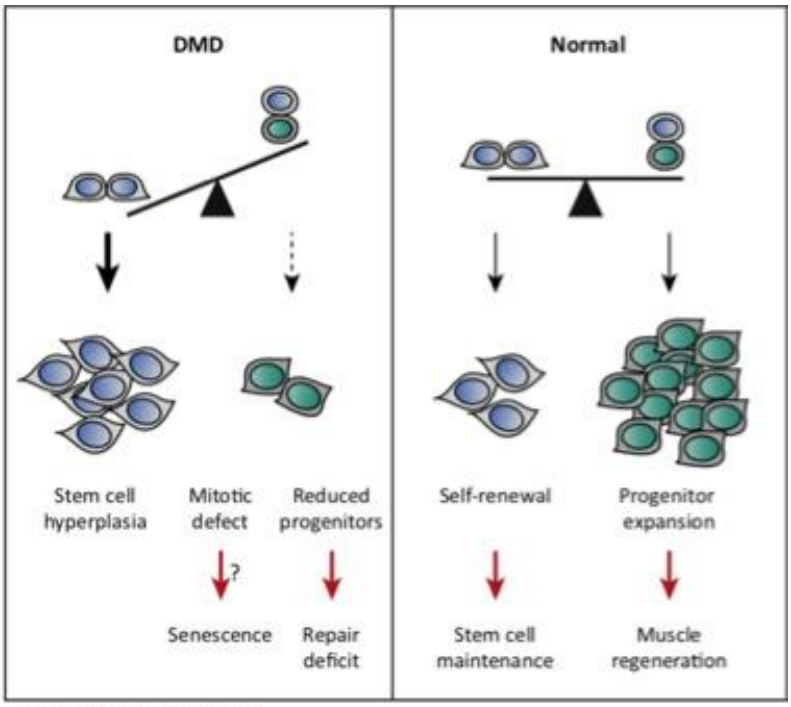
Source: Satellos Biosciences Inc.

As shown in the following image, satellite cells can divide via two mechanisms: symmetric division (self-renewal) in which both daughter cells are identical satellite cells, and asymmetric division in which one satellite cell and one progenitor cell are produced (Chang *et al.*, 2016). In addition, once produced, progenitor cells can go through a finite number of divisions to repair muscle.



Source: Chang *et al.*, 2016

Interestingly, it was reported that *Dmd*, the gene that encodes dystrophin, is expressed in satellite cells, which was seen using both RNA-Seq to identify the *Dmd* transcript and immunofluorescence to identify the dystrophin protein (Dumont *et al.*, 2015). Dystrophin is known to associate with the cell polarity regulating protein MARK2 (Yamashita *et al.*, 2010) and cell polarity proteins are implicated in asymmetric cell division of satellite cells (Troy *et al.*, 2012). In the *mdx* mouse model, satellite cells lack dystrophin and have decreased expression of MARK2, which leads to the loss of polarization and a significant reduction in asymmetric cell division. It is this lack of asymmetric cell division that leads to a gradual loss of myogenic progenitor cells and impaired muscle regeneration, which is shown in the following image. These results indicate that DMD may be due at least in part to a deficiency in muscle stem cell division and suggests that targeting stem cells may prove to be beneficial for DMD patients.



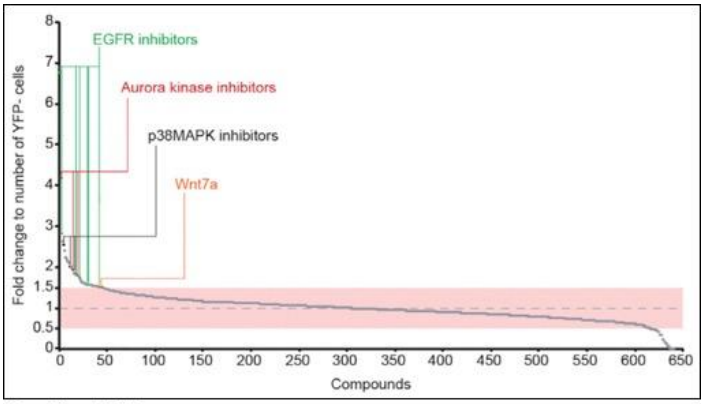
Source: Chang *et al.*, 2016

Targeting Muscle Stem Cell Division for Treating DMD

DMD patients have a dystrophin deficiency in skeletal muscle cells that makes them susceptible to damage (Cohn *et al*., 2000), however a deficit of dystrophin in muscle stem cells is also a part of the mechanism by which DMD patients experience progressive skeletal muscle wasting (Dumont *et al*., 2015). The loss of dystrophin in muscle stem cells results in a loss of polarity in those cells and ultimately a decrease in asymmetric cell divisions, myogenic progenitors, and ultimately the degeneration of myofibers and replacement with adipose and fibrotic tissue.

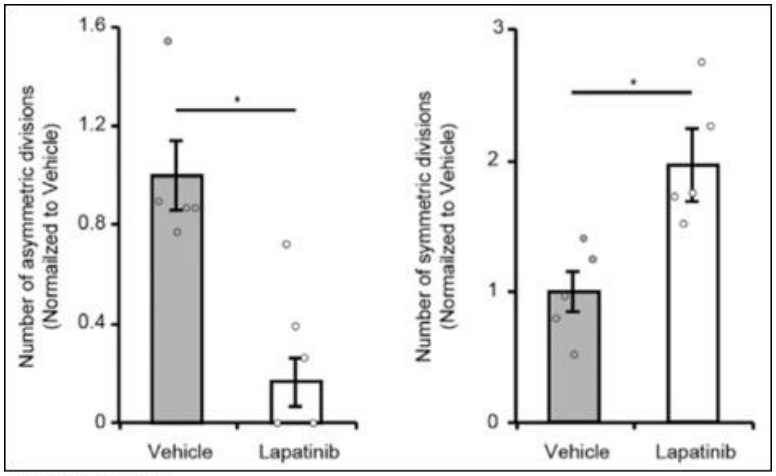
The current approaches to treating DMD patients are all less than ideal, thus a novel mechanism of action to target is desperately needed, particularly one that would be applicable to all DMD patients regardless of mutational status. Recently, Satellos scientists identified novel signaling pathways that are implicated in asymmetric cellular division that could be therapeutically exploited. While the following results describe the work with the epidermal growth factor receptor (EGFR) signaling pathway, the company has identified additional signaling pathways that may be just as, if not more, amenable to therapeutic intervention. However, the work with the EGFR signaling pathway represents a clear proof-of-concept for the company's strategy of using small molecule therapeutics for treating muscle degenerative diseases.

A small molecule screen was performed with 640 well characterized pharmacological compounds to determine if any stimulated satellite stem cell expansion using myofibers isolated from *flexor digitorum brevis* (FDB) muscles, with Wnt7a utilized as a positive control (Wang *et al*., 2019). Forty-three candidate compounds were identified that induced satellite stem cell expansion, as shown in the following image. While Wnt7a was utilized as a positive control, p38MAPK signaling is known to drive satellite cell commitment (Bernet *et al*., 2014), thus the identification of compounds that inhibit the p38MAPK pathway was further validation that the assay was capable of identifying compounds that drive satellite stem cell expansion. Lapatinib, an FDA approved EGFR-Erbb2 inhibitor, was identified as a lead compound along with ZM 449829, another inhibitor of EGFR (Brown *et al*., 2000). Microarray data showed that *Egfr*, *Aurka*, and *Aurkb* are all expressed in satellite cells and are the likely targets of the inhibitors in the screen.



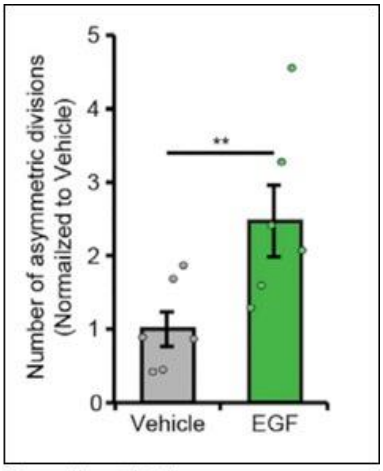
Source: Wang et al., 2019

To confirm the activity of the lead compounds and show it is not specific to FDB muscle satellite cells, myofibers from *extensor digitorum longus* (EDL) muscles were cultured. Treatment of these cells with lapatinib resulted in a shift toward symmetric stem cell division, which included an 83% decrease in the number of asymmetric divisions observed and a 71% increase in the number of satellite stem cells, as shown in the two figures below. There was no change in the total number of satellite cells due to EGFR inhibition, thus indicating it does not alter proliferation.



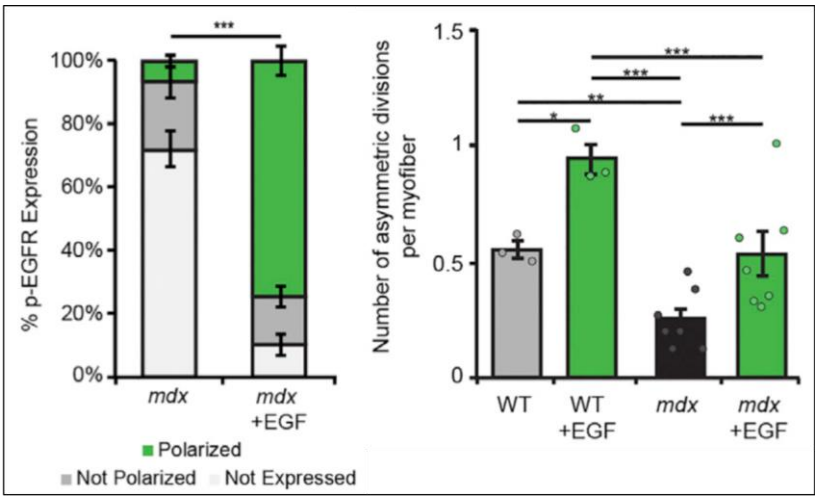
Source: Wang et al., 2019

While EGFR inhibition resulted in a decrease in asymmetric cell division and an increase in symmetric division, addition of EGF to growth medium resulted in an increase in the number of asymmetric divisions compared to vehicle, as shown in the following image.



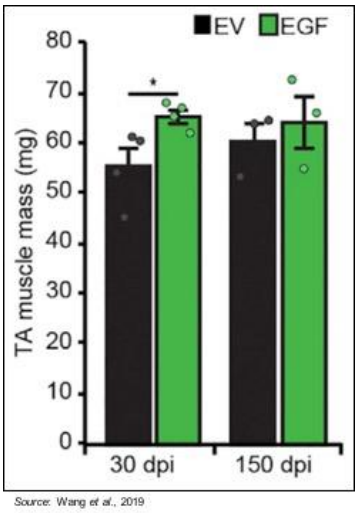
Source: Wang et al., 2019

How EGFR signaling functions in satellite stem cells without dystrophin was examined in EDL myofibers isolated from *mdx* mice. Following one hour of EGF stimulation, the cells were stained for phosphorylated EGFR (a sign of activation). The following graph on the left shows that EGFR was stimulated (with a significant increase in the percentage of polarized cells), thus indicating it retains normal activity even if dystrophin is not present. The following graph on the right shows that EGF is able to restore the number of asymmetric divisions in *mdx* mice to levels identical to the untreated wildtype mice, thus showing the potential for augmenting the EGFR pathway to restore normal muscle stem cell function.



Source: Wang *et al.*, 2019

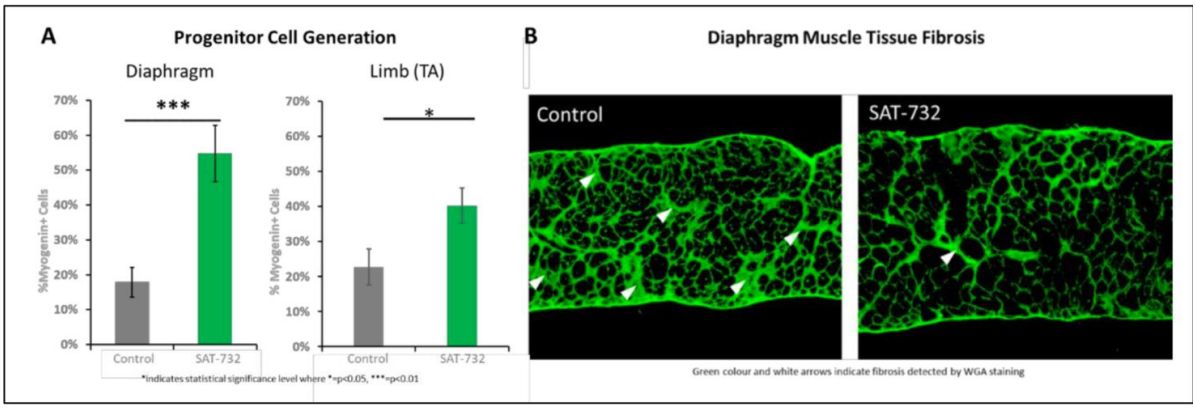
Since DMD is a long-term, progressive muscle disease, the long-term effect of EGF treatment on *mdx* cells was studied through electroporation of TA muscles in *mdx* mice with an EGF expression vector. At both 30- and 150-days post-intervention (dpi), muscles electroporated with the EGF expression vector showed an increase in muscle mass, as shown in the following image. In addition, there was an increase in the total number of myofibers and an increase in force in the EGF expression vector electroporated muscles.



Source: Wang *et al.*, 2019

These results indicate that stimulating the EGFR signaling pathway results in the long-term augmentation of muscle strength in *mdx* mice through increased asymmetric muscle stem cell division, an improvement in regeneration potential in muscle stem cells, and decreased disease progression in the *mdx* mouse model of DMD. Importantly, these effects are seen even in the absence of dystrophin. Thus, the EGFR signaling pathway could represent a novel target for pharmaceutical intervention in the treatment of DMD patients.

Subsequent to the Wang *et al*. publication Satellos identified a specific enzyme in the EGFR pathway ("PTPX") as an initial target for drug discovery efforts. Ultimately, the company identified a compound ("SAT-732") that served as a small molecule inhibitor of PTPX. SAT-732 was shown to restore muscle regeneration capacity and muscle strength to near normal levels in *Mdx* mice. The following image on the left shows the change in diaphragm and limb muscle following treatment with SAT-732, while the images on the right are representative slides showing a statistically significant increase in muscle diameter and reduced fibrotic tissue markers in diaphragm tissue.



Source: Satellos Bioscience, Inc.

Following the generation of initial proof-of-concept data with SAT-732, Satellos has designed and evaluated hundreds of compounds through lead optimization work and has prioritized two chemical families. Additional work is ongoing to optimize the pharmacokinetic properties of several compounds, with additional improvements required before developing an effective inhibitor of PTPX.

In addition to focusing on the PTPX pathway, Satellos identified multiple signaling pathways that may exert an influence on asymmetric stem cell division and muscle regeneration, with a particular focus on the Notch pathway. Satellos has identified a particular protein (nicknamed 'K9') in the Notch pathway that it believes has a previously unrecognized role in muscle cell regeneration. Following results obtained through genetic ablation of K9, the company hypothesizes that targeting that protein could modulate asymmetric muscle stem cell division in a manner similar to PTPX. At the start of 2022, Satellos began directing efforts towards a drug discovery program aimed at identifying inhibitors of K9. Since K9 is already the target of other pharmaceutical interventions in a different disease setting, Satellos was able to synthesize existing K9 inhibitors to begin initial testing of its hypothesis. The company has already created multiple candidate inhibitors of K9 and is continuing to optimize these compounds in search of a lead candidate. We anticipate the K9 program taking precedence over the PTPX program by the end of 2022.

Case Reports of Milder Symptoms in DMD Patients

Targeting muscle stem cells will not change the fact that DMD patients lack muscle dystrophin, which is thought to be the root cause of the disease. However, there are case reports in the medical literature of DMD patients with a very mild form of the disease even though they have mutations that result in little to no dystrophin expression. These reports support the notion that a patient can have functional muscles even with little to no dystrophin.

Zatz *et al*. describe two case studies of patients with DMD and very mild phenotypes (Zatz *et al*., 2014). The first case study involved two half-brothers. One brother was diagnosed with DMD at age 7 and was wheel-chair bound at age 9. The other brother had some muscle weakness at age 13, however as of age 15 (when the report came out) was only mildly affected with some difficulty running but normal walking. Dystrophin levels in both brothers was <5% of normal and despite both of them having a similar lack of dystrophin they had very different phenotypes.

The second case study involved a 16-year-old who has an out-of-frame deletion in the dystrophin gene and was negative for dystrophin by immunostaining. In spite of this, the patient is doing well with discrete calf hypertrophy and a normal stride.

Mild DMD phenotypes are also seen in animal models of the disease. The model with the most similarity to the human condition is the golden retriever muscular dystrophy (GRMD) dog (Cooper *et al*., 1988). These dogs have progressive muscle degeneration, atrophy, fibrosis, and elevated serum creatine kinase (CK), with death typically occurring at around 1-2 years of age. In a Brazilian research center, two dogs were reported on that had a very mild phenotype despite the absence of muscle dystrophin (Zucconi *et al*., 2010). The dogs remained fully ambulatory and had normal lifespans in spite of the fact they had no muscle dystrophin, elevated serum CK, and no sign of utrophin upregulation. Further analysis of these dogs led to the identification of candidate genomic region that associated with the mild phenotype. This genomic region contained a muscle-specific transcription factor binding site that drives the overexpression of *Jagged1*, a protein that functions in the Notch pathway (Vieira *et al*. 2015). The dogs with increased *Jagged1* expression had a greater proliferative capacity of their myoblasts, thus suggesting *Jagged1* could be involved in muscle cell proliferation and repair.

The important point from these case studies is that they show it is possible to have functional muscle activity with little to no muscle dystrophin and that signaling pathways exist that can affect muscle cell proliferation and repair in the absence of dystrophin. Thus, this supports the therapeutic strategy focused on restoring proper muscle stem cell division rather than restoring the expression of dystrophin.

GWAS Study Identifies Stem Cell Polarity Gene as Candidate Genetic Modifier

In two published manuscripts, researchers at Ohio State/Nationwide Hospital performed a genome-wide association study (GWAS) that examined 419 DMD patients to identify potential candidate genetic modifier loci based on age at loss of ambulation (Flanigan *et al*., 2021). The researchers limited the dataset to patients with strict 'loss-of-function' mutations in the *DMD* gene to minimize the impact of trace dystrophin expression (Waldrop *et al*., 2017) and applied specific statistical analyses that are well suited to smaller sample sizes.

The analysis identified six candidate loci, with several of them having not been previously identified (Flanigan *et al*., 2021). One of the most interesting of these is single nucleotide polymorphism (SNP) rs2061566 on Chromosome 18, which is located within an enhancer that regulates the par-6 family cell polarity regulator gamma (*PARD6G*) gene. PARD6G is a member of the PAR polarity complex that participates in the control of asymmetric cell division and self-renewal of muscle satellite cells (Dumont *et al*., 2015). The authors posit that an increased focus on cell polarity in DMD patients is warranted, and that micro-dystrophin gene therapy "will not correct fundamental muscle stem cell defects important to muscle maintenance". This is a surprising conclusion as the authors previous work is focused on gene therapy treatments for DMD. Most importantly, we believe this provides significant outside validation to the theory that the dysregulation of muscle stem cell renewal is driving DMD and that Satellos is on the right path in trying to correct that dysregulation.

Background on Duchenne Muscular Dystrophy

DMD is an X-linked recessive disorder that is the most common muscular dystrophy, which are a group of diseases that result in progressive weakness and loss of muscle mass. DMD occurs in approximately 1 in 5000 boys and the average age of diagnosis is five years of age (Moat *et al*., 2013).

Patients typically present with gross motor delay, gait abnormalities, and frequent falls. Most boys with DMD will gain strength until approximately six years of age, however after this progressive deterioration in strength will occur and without treatment most will need a wheelchair around age 10 (Falzarano *et al*., 2015). Cardiomyopathy presents in all patients by the age of 18, however most are asymptomatic due to limited physical activity (Nigro *et al*., 1990). Respiratory complications are found in all patients and respiratory failure is the leading cause of death for DMD patients (Phillips *et al*., 2001).

DMD is caused by a mutation in the *DMD* gene, which encodes the dystrophin protein (Hoffman *et al*., 1987). The *DMD* gene contains more than 2.5 million base pairs of the X chromosome (approximately 1.5% of the X chromosome). The coding sequence is 86 exons, which leads to a 14,000 base pair messenger RNA that is predominantly expressed in skeletal and cardiac muscle, with lesser amounts expressed in the brain (Muntoni *et al*., 2003). Three different promoters give rise to different full-length isoforms, with additional isoforms derived from alternative splicing events (Sadoulet-Puccio *et al*., 1996).

The dystrophin protein is 427 kDa and comprises four domains. It is associated with the plasma membrane of cardiac and skeletal muscle (sarcolemma) where it interacts with various integral membrane proteins that together form the dystrophin-glycoprotein complex (DGC). The main role of the DGC is to stabilize the muscle fibers during contractions as well as propagate cell survival and cellular defense signaling pathways (Rando 2001).

Mutations in the *DMD* gene include intragenic deletions (60-65%), duplications (5-15%), and various combinations of point mutations, intronic deletions, and exonic insertions. While deletions can happen anywhere in the gene, two "hotspots" exist within exons 2-19 toward the 5' end of the gene and exons 45-55 in the middle of the gene (Muntoni *et al*., 2003). There is no direct correlation between the size of the deletion in the *DMD* gene and disease severity, with the phenotype for DMD patients being dependent upon whether the mutation disrupts the reading frame.

The most widely used animal model of DMD is the *mdx* mouse, which has a point mutation in exon 23 of the *mdx* gene that prevents the production of the dystrophin protein (Sicinski *et al*., 1989). These animals exhibit a number of similar phenotypes as DMD patients, including elevated CK levels, irregular muscle histology and necrosis, decreased respiratory function with age, and multiple cardiac phenotypes, including myocardial fibrosis by 6 months of age (Stuckey *et al*., 2012).

Therapeutic Options for DMD

There is currently no cure for DMD, and all therapeutic approaches are designed to manage symptoms, treat expected complications, and improve a patient's quality of life as much as possible. Multiple advances over the past 30 years has resulted in an increase in expected survival time, with standards of care for DMD patients published in 2010 (Bushby *et al*., 2010).

Standard of care for DMD patients begins with corticosteroids. Multiple short-term and long-term studies have shown that their use leads to muscle strength improvement across a number of parameters (Matthews *et al*., 2016). In addition, corticosteroid therapy has been shown to increase ambulation by approximately three years (Biggar *et al*., 2006). Emflaza® (deflazacort) was approved by the FDA for the treatment of DMD in 2017 and is currently approved to treat DMD patients 2 years of age and older, regardless of mutation status. In 2021, Emflaza generated revenues of $187 million for PTC Therapeutics (EvaluatePharam).

While offering a number of positive benefits to patients, corticosteroid therapy results in a number of potentially serious side effects that must be effectively managed. These side effects include excessive weight gain, gastric complications, cataracts, hypertension, behavioral changes, bone fracture, and growth suppression (Bushby *et al*., 2010).

The adverse effects of corticosteroids are hypothesized to be caused by transactivation of downstream signaling pathways following binding to the glucocorticoid receptor. Vamorolone is a compound that binds to the same receptors as corticosteroids but without the transactivation effects. It is currently in a Phase 2b trial following positive results from a Phase 2a study in which vamorolone exhibited an improved side effect profile compared to glucocorticoids (Conklin *et al*., 2018).

Newer therapies developed for DMD include gene addition, exon skipping, stop codon readthrough, and genome editing techniques, which are all designed to try to restore some level of dystrophin expression.

- Gene Addition: The idea behind gene therapy is that if a functional copy of the *DMD* gene can be inserted into patients' cells it may help to restore muscle function. Adeno-associated virus (AAV) is the only viral vector that will infect muscle, however its genome is relatively small (~4.5 kb) and the full-length *DMD* mRNA is very large (14 kb). As a workaround, researchers discovered that as long as two domains of dystrophin are present, the actin-binding domain at the N-terminus and the dystroglycan binding domain at the C-terminus, the protein retains at least partial activity. Thus, multiple 'microdystrophins' have been designed that can fit inside the AAV vector.

  SRP-9001: This microdystrophin is being developed by Sarepta Therapeutics. In January 2021, the company reported results from a Phase 2 clinical trial in which there was no statistically significant difference in functional motor ability between patients treated with SRP-9001 and placebo after 48 weeks. However, the company has performed multiple additional analyses of the data from three different studies of SRP-9001 and compared results to external control data, with those results showing what Sarepta believes are statistically significant and clinically meaningful benefits. In September 2022, Sarepta announced it was filing a Biologics License Application for SRP-9001. The company has proposed the ongoing Phase 3 EMBARK clinical trial as a post-marketing confirmatory study.

  PF-06939926: This microdystrophin is being developed by Pfizer. It is currently being tested in a Phase 3 clinical trial following positive results reported in a Phase 1b trial. In December 2021, Pfizer announced that the trial was put on a clinical hold following the death of a patient. There were no details surrounding the patient's death, however it does point to the inherent risk of gene therapy using AAV (see below).

  While the idea of adding a functional microdystrophin through gene delivery is an intriguing one, the strategy does have a few drawbacks. First, the human body has over 500 muscles, thus it is very difficult to get viral infection of all them. Second, since AAV is episomal, normal muscle cell turnover will result in loss of the transgene over time. Third, an immune response can be developed to AAV, the microdystrophin, or both. Fourth, the manufacturing of the viral vector is quite time consuming and improvements in large scale manufacturing of AAV will be necessary in order to treat a large number of patients.

  Lastly, the FDA recently held a workshop to discuss safety concerns surrounding the use of AAV, as the agency noted that approximately one-third of recent AAV gene therapy trials had a treatment-emergent serious adverse event. For example:
  - A clinical trial in 2020 of an Audentes Therapeutics gene therapy lead to severe liver-related side effects and the deaths of three patients.
  - Multiple patients in Solid Biosciences' Phase 1/2 clinical trial of its DMD therapy experienced immune-related side effects, including acute kidney injury and complement activation.
  - Pfizer recently limited enrollment in a clinical trial of its DMD gene therapy following three cases of muscle weakness, two of which involved myocarditis.

  A DMD treatment that can avoid the use of AAV would be advantageous, as it would prevent any of the above-mentioned complications.

- Exon Skipping: In patients that harbor a mutation in the *DMD* gene that disrupts the reading frame of the dystrophin mRNA, exon skipping is utilized in an effort to prevent the exon carrying the mutation from being included in the final mRNA. Four antisense oligonucleotides (ASO) have been approved that target mutations in three different exons.

  Exondys 51 (eteplirsen): Approved in 2016, this treatment targets mutations in exon 51, which covers approximately 14% of DMD patients. It generated $454 million in revenues for Sarepta Therapeutics in 2021 (EvaluatePharma).

Vyondys 53 (golodirsen): Approved in 2019 and targets mutations in exon 53, which covers approximately 8% of DMD patients. It generated $90 million in revenues for Sarepta Therapeutics in 2021 and is estimated to generate $233 million in revenues in 2026 (EvaluatePharam).
Viltepso (viltolarsen): Approved in 2020 and targets mutations in exon 53. It generated $78 million in revenues for NS Pharma in 2021 and is estimated to generate $429 million in revenues in 2026 (EvaluatePharma).
Amondys 45 (casimersen): Approved in 2021 and targets mutations in exon 45, which covers approximately 8% of DMD patients. It is estimated to generate $246 million in revenues in 2026 (EvaluatePharma).

Exon skipping can result in expression of a truncated dystrophin, however given the turnover of the ASO it is necessary to continually dose patients. In addition, it is very difficult to achieve expression of the truncated dystrophin in all muscle cells in the body. Given the specificity of each compound, only a certain percentage of DMD patients can be treated effectively by each drug.

- Stop Codon Readthrough: In patients with a mutation in the DMD gene that results in a premature stop codon (nonsense mutations), stop codon readthrough is an option to bypass the stop codon and produce a functional dystrophin. The field began in 1979 where stop codon readthrough was reported for aminoglycosides (Palmer *et al*., 1979). The aminoglycoside antibiotic gentamicin was tested in DMD patients and was shown to significantly increase the level of dystrophin to 15% of normal (Malik *et al*., 2010).

  Translarna (ataluren): This compound was approved for the treatment of DMD by the E.U. in 2014. It generated $236 million in revenues for PTC Therapeutics in 2021 and is forecast to generate $352 million in 2026 (EvaluatePharma).

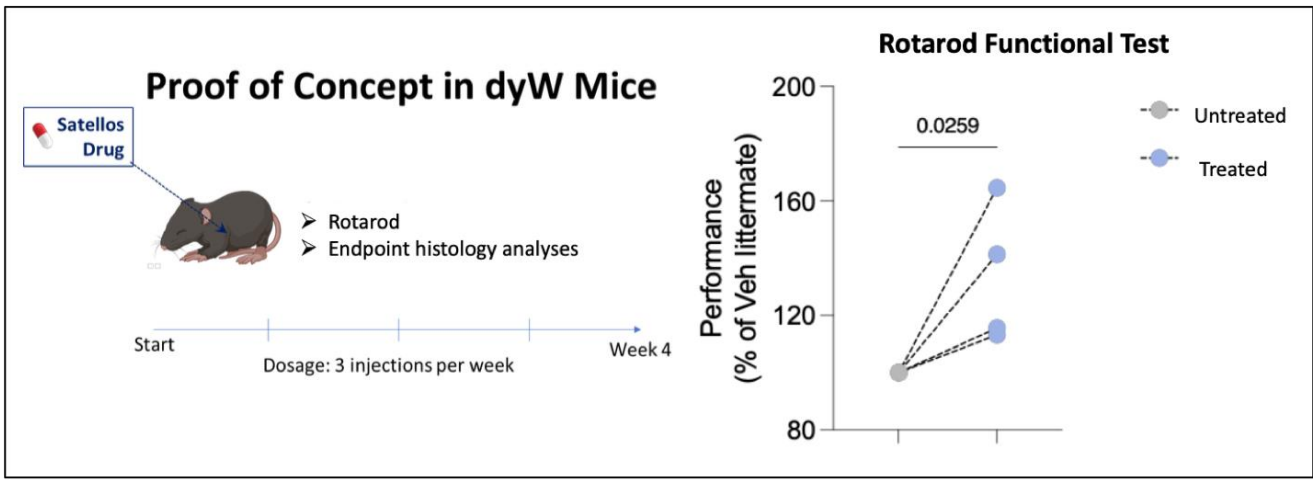
- Genome Editing Techniques: CRISPR-Cas9 technology can be utilized to incur double stranded DNA breaks at specific places in the genome. DNA repair can initiate homologous recombination (in dividing cells) or non-homologous end joining (in non-dividing cells). For non-dividing cells such as muscle, non-homologous end joining can be used to delete exons with mutations to restore the reading frame of the *DMD* gene.

Additional Disease Indications

In addition to DMD, Satellos believes that the company's technology will be applicable to other muscular dystrophies. While the other muscular dystrophies each have a unique cause, and can result in conditions ranging from benign to severe, Satellos will be focusing on a subgroup of dystrophies associated with the DGC. The company has identified multiple forms of muscular dystrophy that could potentially be treated via the same mechanism of action as DMD. In January 2022, Satellos announced a sponsored research agreement with the Université de Sherbrooke to evaluate candidate drug molecules in disease models of rare and ultra-rare orphan dystrophies, including:

Lama2-Related Muscular Dystrophies (LAMA2-RD): These are autosomal recessive disorders caused by mutations in the *LAMA2* gene, which encodes for the alpha 2 subunit of the heterotrimeric Laminin-2 protein (Lm-211). Lm-211 is a component of the extracellular matrix of skeletal muscle, Schwann cells, placental trophoblast, dermis-epidermis junction, and myocardium (Vainzof *et al*., 2005). Patients with the disease can suffer from a range of phenotypes, including a milder, later childhood-onset limb-girdle muscular dystrophy to a severe, early-onset congenital muscular dystrophy. The prevalence of LAMA2-RD is approximately 0.6-0.7/100,000 (Norwood *et al*., 2009).

On October 11, 2022, Satellos announced a poster presentation at the World Muscle Society Congress that included preliminary results showing small molecule targeting of cell polarity leads to an increase in rotarod functional test in a mouse model of LAMA2-RD (dyW mice), as shown in the following figure. These early results may indicate that Satellos' technology may be applicable to diseases other than DMD.



Source: Mitchell, 2022

Collagen-VI Related Muscular Dystrophies (COL6-RDs): These are a group of disorders caused by mutations in any one of the three collagen VI genes (*COL6A1*, *COL6A2*, *COL6A3*). Collagen VI is a component of the extracellular matrix in muscle, skin, tendon, and vessels. The COL6-RDs result in a range of phenotypes, including the more severe Ullrich congenital muscular dystrophy (UCMD) down to the less severe Bethlem myopathy (BM). The prevalence of UCMD was reported as 0.13/100,000 and for BM it was 0.77/100,000 (Norwood *et al*., 2009).

Neither of those indications currently has effective treatment options and would likely offer the opportunity for rapid clinical trial readouts. In addition, both programs would likely be available for orphan and rare pediatric disease designations and could potentially be eligible for non-dilutive funding. Rare Pediatric Disease designation includes the potential for receiving a priority review voucher (PRV) upon approval of the drug. The PRV is fully transferable and multiple PRVs have been sold recently for approximately $100 million each.

Intellectual Property

Satellos holds a number of issued and pending patents related to its discoveries in muscle stem cell biology and potential experimental and therapeutic applications, which comprise four patent families. These patents and applications are licensed from the Ottawa Hospital Research Institute (OHRI), where Dr. Rudnicki's research lab is located.

PCT/US2018/027920 – A method of stimulating asymmetric division of satellite stem cells. These cover the EGFR/PTPX drug development program.

PCT/US2012/055396 – Wnt7a compositions and methods of using the same. These cover the Wnt7a drug development program.

PCT/CA2010/000601 – Compositions and methods for modulating stem cells and uses thereof. These cover the Wnt7a drug development program.

63/345/678 – Modulation of satellite cell polarity and asymmetric cell division. These cover the Notch/K9 drug development program.

The company has also recently arranged for the drafting and filing of patent applications related to drug discovery targets originating in Dr. Rudnicki's lab, titled "Modulation of satellite cell polarity and asymmetric cell division" (derived from 63/355,903). This application will be added to the license agreement between Satellos and OHRI in the near future.

Financials and Capital Structure

In August 2022, Satellos announce financial results for the second quarter of 2022. The company did not have any revenues in the second quarter of 2022 compared to CAD$50,000 in grant income for the second quarter of 2021, which was received from The Foundation for Gene & Cell Therapy – Jesse's Journey. Net loss for the second quarter of 2022 was CAD$2.2 million compared to CAD$0.9 million for the second quarter of 2021. Expenses consisted of:

- R&D expenses net of refundable tax credits was approximately CAD$0.6 million in the second quarter of 2022 compared to approximately CAD$0.3 million for the second quarter of 2021. The increase was due to the company ramping up R&D contractor spending.
- Management fees and salaries was approximately CAD$0.4 million for the second quarter of 2022 compared to approximately CAD$0.2 million for the second quarter of 2021. The increase was due to increased salaries and consulting fees.
- Professional fees were approximately CAD$0.5 million for the second quarter of 2022 compared to approximately CAD$0.3 million for the second quarter of 2021. The increase was due to higher spending on accounting, audit, and legal fees due to the company being publicly-traded in the current period.
- G&A expenses were approximately $0.3 million for the second quarter of 2022 compared to CAD$3,497 for the second quarter of 2021. The increase was due to increased depreciation and amortization due to amortization of the technology acquired from iCo therapeutics following the reverse merger.
- Stock-based compensation was approximately CAD$0.4 million for the second quarter of 2022 compared to CAD$15,447 for the second quarter of 2021. The increase was due to a larger number of options granted during the current period.

As of June 30, 2022, Satellos had approximately CAD$1.9 million in cash and cash equivalents. Subsequent to the end of the quarter, the company announced the closing of a public offering in which it sold 8.75 million units at a price of CAD$0.40 per unit, raising gross proceeds of CAD$3.5 million. Each unit consisted of one share of common stock and one-half of one common share purchase warrant with an exercise price of CAD$0.60 and an expiration date of September 13, 2025. As of June 30, 2022, Satellos had approximately 33.0 million shares outstanding and when factoring in stock options, warrants, and the latest financing, we estimate the company a fully diluted share count of approximately 49.4 million.

Risks to Consider

Satellos is a very early stage drug development company with a limited operating history, thus it is difficult to judge how likely it is to succeed in its efforts. There is no assurance that the company will be successful in achieving any of its business objectives or providing a positive return for investors. Some of the potential risks are outlined below, but investors should consult the company's "Management's Discussion and Analysis" for additional risk factors.

Development Risk: Given that Satellos is at a very early stage of the drug development process, there is no assurance that the company will be successful at developing any drug candidates. The drug development process is time intensive and will require numerous preclinical and clinical trials to prove any drug candidate is safe and effective. Drug development is subject to numerous scientific and regulatory unknowns and there can be no assurance that even if a drug is successfully developed that it will be accepted by patients/doctors/payers.

Clinical Risk: The company is currently in the preclinical stage of development. Statistically speaking, drug candidates at this stage of development have a very high probability of failure. Even if the drug is shown to be safe in animal testing, serious adverse effects could be discovered during clinical testing that could delay the development of the drug candidate or lead to discontinuation of its development. Any drug candidate could show efficacy in animal models of a disease but fail to show efficacy in a patient

population. If Satellos is unable to produce a compound that results in positive clinical trial results, the company's business would be severely impacted.

Financing Risk: The drug development requires a significant amount of capital to move a drug candidate from discovery through clinical trials and to approval. Satellos currently has sufficient capital to fund operations into the second quarter of 2023, and the company will require significant additional capital as it moves forward in the drug development process. There can be no assurance that the company will be able to obtain the financing it requires or that it will be able to obtain on terms that are favorable to current shareholders.

MANAGEMENT PROFILES

Frank Gleeson – Chief Executive Officer

Mr. Gleeson has been involved in over 20 biomedical companies and has negotiated numerous financing and M&A transactions valued in excess of $500 million. Prior to founding Satellos in early 2018, he spent four years with the Centre for Probe Development and Commercialization (CPDC) where as Chief-Commercial Operations Mr. Gleeson was instrumental in building a global radiopharmaceutical manufacturing business and supporting the creation of two spin-out companies. Prior to CPDC, he served as an Executive-in-Residence with the Fight Against Cancer Innovation Trust (FACIT), an innovative nucleator, where he supported or led the creation, financing, and exits of 3 new entities. In 2008 Mr. Gleeson co-founded, with Dr. Rudnicki, a stem-cell biotechnology company which they successfully merged into Fate Therapeutics Inc. (NASDAQ: FATE) in 2010. Earlier, Mr. Gleeson was founding CEO of MDS Proteomics Inc., a strategic corporate play focused on mapping the proteome, where he made and integrated 3 acquisitions, built leading-edge sequencing infrastructure, a 200-person team, and raised in excess of $100M. From 1994-2000, Mr. Gleeson was a senior vice president and venture partner with MDS Capital Corp. (now Lumira), where he was lead partner on a fund with over $250M under management focused on creating drug discovery companies based on novel Canadian science. Prior to his tenure with MDS, Frank enjoyed a 17-year operational career with ICI plc (now AstraZeneca), a global chemicals, pharmaceuticals and advanced materials company, during which he was involved in technology commercialization in several fields both in Canada and internationally. Mr. Gleeson has served on numerous Boards of private and public companies and not-for profit entities. He is a member of the investment advisory committee of Fighting Blindness Canada.

Michael Rudnicki, PhD – Chief Scientific Officer

In a landmark 2007 *Cell* paper, Dr. Rudnicki was the first to define so called muscle stem cells (a.k.a. 'satellite cells') and characterize a subpopulation as bona fide multipotent stem cells capable of both self-renewal and regeneration. Building on this seminal work, Dr. Rudnicki has established a continuous record of foundational findings and discoveries spanning a 25-year research career. In so doing, he has transformed the field's understanding of the nature and role muscle stem cells play in the lifelong repair and growth processes of skeletal muscle, the body's largest organ. Of extraordinary significance, Dr. Rudnicki and his team made the breakthrough identification of a signaling role for dystrophin in orienting muscle stem cells to divide asymmetrically to produce muscle progenitor cells. This seminal discovery transforms the classical understanding of Duchenne muscle dystrophy as a disease of muscle fragility to a new paradigm in which defective muscle regeneration can be seen as a root cause of the progressive muscle loss experienced by patients living with Duchenne. These findings offer new hope for the development of an entirely novel potential therapeutic approach to treating Duchenne and possibly other incurable and fatal muscle disorders. Dr. Rudnicki is the Scientific Director of the Canadian Stem Cell Network, Director of the Regenerative Medicine Program at the Ottawa Hospital Research Institute, a Professor in the Faculty of Medicine at the University of Ottawa, and an Associate Editor of the Journal of Cell Biology as well as Cell Stem Cell. He has published 235 scientific articles, authored 14 patents, with an hindex of 96 based on 44,000+ citations of his work. Dr. Rudnicki's contributions have been recognized with numerous honors including being named a Tier 1 Canada Research Chair, a Fellow of both the Royal Society and the Royal Society of Canada, a past International Research Scholar of the Howard Hughes Medical Institute for the maximum allowable two consecutive terms, and an Officer of the Order of Canada. He received his Ph.D. at the University of Ottawa and trained at the postdoctoral level at the Massachusetts Institute of Technology in the Whitehead Institute under Dr. Rudolf Jaenisch.

Warren Whitehead – Chief Financial Officer

Mr. Whitehead is a Chartered Professional Accountant (CPA) and Certified Management Accountant (CMA) who has held multiple senior financial management positions in several biotechnology and pharmaceutical companies, and is currently a Board Member of Aptose Biosciences. Mr. Whitehead was

© Copyright 2022, Zacks Investment Research. All Rights Reserved.

the former Chairman and Board Member of Plantform Corporation and a former Board Member of Telesta Therapeutics (TSX), which was acquired by Prometic Life Sciences in 2016. He served as the Chief Financial Officer of ProMIS Neurosciences Inc. (formerly Amorfix Life Sciences Ltd.) from 2013 to 2015, a TSX-listed company targeting detection and effective treatment of Alzheimer's disease and amyotrophic lateral sclerosis. Previously, from 2006 to 2008, he was the Chief Financial Officer of Arius Research Inc., a TSX-listed company developing anti-cancer antibodies, where he provided financial guidance and leadership during the acquisition of Arius by Roche in 2008. He was also the former Chief Financial Officer of Labopharm Inc. from 2000 to 2006, where he completed a series of public equity financings, including a cross-border Nasdaq offering. Other positions include Chief Financial Officer of Resolution Pharmaceuticals Inc., and a position in finance and business development at Glaxo Canada (now GlaxoSmithKline). Mr. Whitehead holds an MBA and BComm from the University of Windsor and a BA from the University of Western Ontario.

Phil Lambert, PhD – Chief Technology Officer

Dr. Lamber is a neuroscientist by training and a high-performing preclinical executive with over twenty-five years of drug discovery and development expertise in the areas of neuroscience, ocular, metabolic, inflammatory and orphan diseases. Phil is an experienced drug finder who has provided the necessary preclinical data to support the entrance of more than twenty small and large molecule therapeutics into the clinic for multiple diseases. He is a serial entrepreneur, who was a member of the management team which conducted the due diligence and sale of Sirtris to GSK in 2008 and co-founded, effectively grew and sold VivoPath, a contract research organization to Charles River Laboratories in 2014. An accomplished leader and a well published researcher, with a unique background having held senior positions in academia, biotechnology, non-profit, large pharmaceutical and contract research organizations Phil has held positions at Centogene, Life Biosciences, Sirtris Pharmaceuticals, Forum Pharmaceuticals, ALS Therapy Development Institute, Regeneron Pharmaceuticals, GSK and Parke Davis. Phil received his Ph.D. in neuroendocrinology from Imperial College, University of London and was a faculty member in the Department of Psychiatry at Emory University School of Medicine.

VALUATION

We are initiating coverage of Satellos Biosciences Inc. (MSCLF) with a $0.70 valuation. Satellos is a biopharmaceutical company developing treatments for muscle diseases, including muscular dystrophies and muscle loss associated with chronic illness and aging. Satellos' core technology is based on discoveries by its scientific co-founder that implicates impairments in muscle stem cell division as a cause of muscle-related diseases. The company has already identified multiple pathways in muscle stem cells that can be targeted for therapeutic intervention and drug discovery efforts are currently underway. The lead indication for Satellos is Duchenne muscular dystrophy (DMD), a rare deadly genetic disease that has limited treatment options, none of which are curative. Proof-of-concept studies in a DMD mouse model showed that targeting muscle stem cells resulted in enhanced muscle quality, performance, and a return to cell division equilibrium. While DMD is the current focus, the company's technology will likely be amenable to treating multiple other conditions, including orphan muscular dystrophies and other muscle wasting conditions.

Advancing Multiple Candidates Targeting Protein in Notch Pathway

Satellos recently announced that it has selected a group of novel small molecule drug candidates to advance into further preclinical studies. These molecules target a particular kinase protein, codenamed "K9" by the company, in the Notch signaling pathway. Inhibiting this protein modulates muscle stem cell division and regeneration. The company will now evaluate and prioritize these candidate molecules for absorption, distribution, metabolism, and excretion (ADME), pharmacokinetics (PK), and *in vivo* efficacy. We anticipate the choice of a lead and back-up candidates for IND-enabling studies to be completed before the end of 2022. IND-enabling studies would then take place during 2023 and an IND could potentially be filed at the end of 2023, with initial clinical studies initiating in early 2024.

Valuation

We value Satellos based on the potential for the company's K9 program in the treatment of DMD. Investors should be aware that Satellos is at a very early stage in the drug discovery process, which means that there will be many adjustments to our model as the company prioritizes a drug candidate molecule and moves it through preclinical testing and into the clinic. Thus, the estimates we provide below are based on the information that is available as of the date of this report and are likely to change multiple times as we learn more information about the lead drug candidate.

Based on the timelines provided above, we estimate that the company will have preliminary Phase 1 data in late 2024 and Phase 2 data in 2026. Given the large unmet need in this population, positive results from a moderately sized Phase 2 trial could be enough to gain accelerated approval, with the FDA likely mandating a confirmatory post-approval Phase 3 trial. Under this scenario we estimate approval in 2027 and revenues to begin in 2028. Investors should be aware this is a best-case scenario and there are a number of factors that could impact this timeline. We estimate peak sales of $1.7 billion in 2033, which we believe is reasonable given that the treatment should not be restricted to a certain mutation status and the potential for disease modification. We apply a 4x multiple, a 5% probability of approval, and a 20% discount rate, which leads to a net present value of approximately $46 million.

Combining the net present value for the K9 program along with the company's current cash position and potential cash from warrant exercises leads to a valuation of approximately $52 million. Satellos currently has a fully diluted share count of approximately 49.4 million and we add 25 million shares for future dilution as the company is very early in the development process. This leads to a current valuation of $0.70 per share.

© Copyright 2022, Zacks Investment Research. All Rights Reserved.

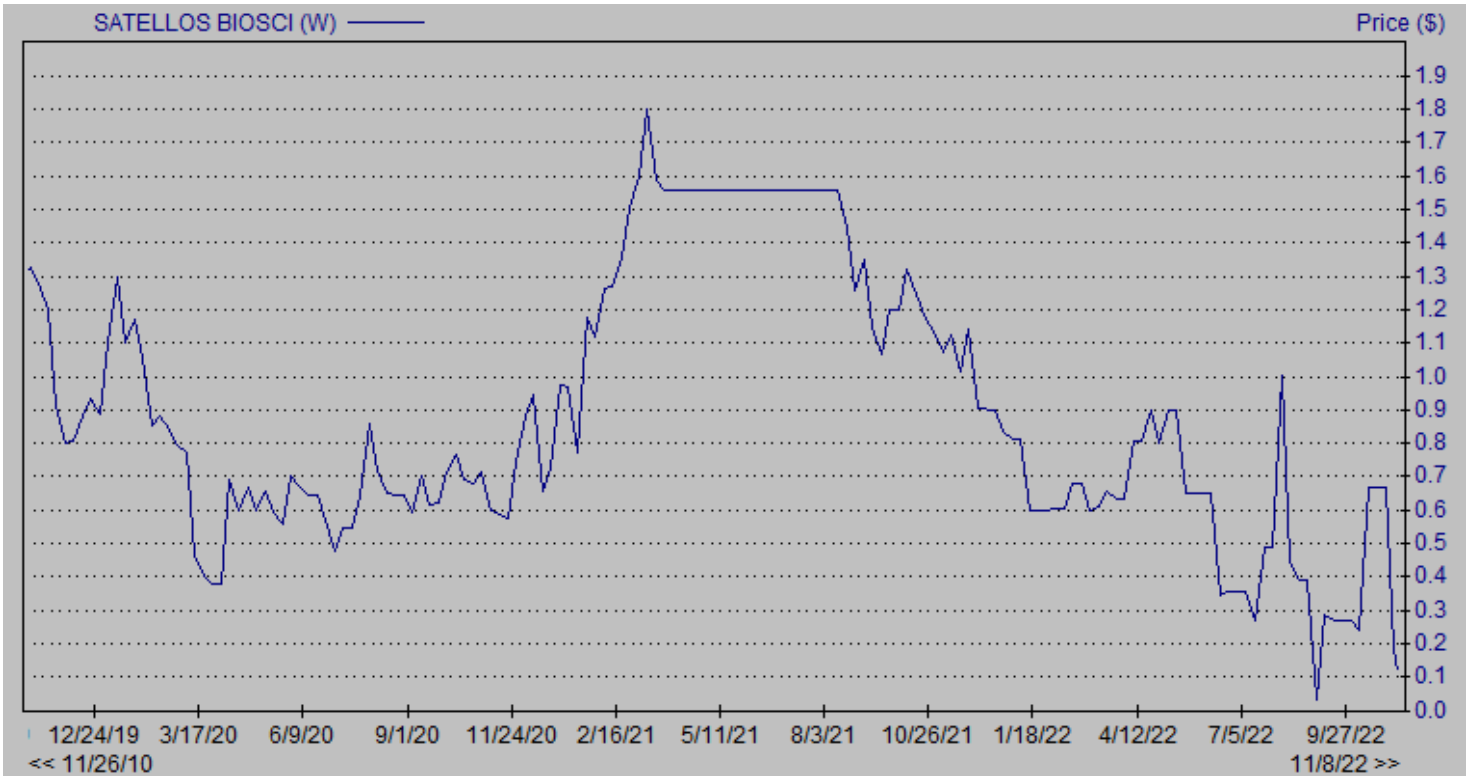
PROJECTED FINANCIALS

Satellos Biosciences Inc. (in $USD)	2021 A	Q1 A	Q2 A	Q3 E	Q4 E	2022 E	2023 E	2024 E
DMD Program	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0
Additional Indication	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0
Other Income	$0.1	$0.1	$0.0	$0.0	$0.0	$0.1	$0.0	$0.0
Total Revenues	**$0.1**	**$0.1**	**$0.0**	**$0.0**	**$0.0**	**$0.1**	**$0.0**	**$0.0**
CoGS	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0
R&D	$1.6	$0.6	$0.5	$0.6	$0.6	$2.2	$2.8	$4.0
SG&A	$1.3	$0.6	$0.8	$0.8	$0.8	$3.1	$3.2	$3.3
Stock-based compensation	$0.8	$0.4	$0.3	$0.4	$0.4	$1.5	$1.6	$1.8
Depreciation and Amortization	$0.1	$0.1	$0.1	$0.1	$0.1	$0.3	$0.3	$0.3
Other expenses	$0.1	$0.0	$0.0	$0.0	$0.0	$0.1	$0.1	$0.1
Operating Income	($3.8)	($1.7)	($1.8)	($1.8)	($1.8)	($7.1)	($7.9)	($9.5)
Operating Margin	-	-	-	-	-	-	-	-
Interest & Other Income	($8.5)	$0.0	$0.0	$0.0	$0.0	($0.0)	$0.1	$0.1
Pre-Tax Income	($12.2)	($1.7)	($1.8)	($1.8)	($1.8)	($7.1)	($7.8)	($9.4)
Taxes & Other	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0
Tax Rate	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*
Net Income	($12.2)	($1.7)	($1.8)	($1.8)	($1.8)	($7.1)	($7.8)	($9.4)
Reported EPS	**($0.51)**	**($0.05)**	**($0.05)**	**($0.05)**	**($0.04)**	**($0.20)**	**($0.13)**	**($0.13)**
Weighted Shares Outstanding	24.1	33.0	33.0	36.0	42.0	36.0	60.0	70.0

Source: Zacks Investment Research, Inc. *David Bautz, PhD*

© Copyright 2022, Zacks Investment Research. All Rights Reserved.

HISTORICAL STOCK PRICE



© Copyright 2022, Zacks Investment Research. All Rights Reserved.

DISCLOSURES

The following disclosures relate to relationships between Zacks Small-Cap Research ("Zacks SCR"), a division of Zacks Investment Research ("ZIR"), and the issuers covered by the Zacks SCR Analysts in the Small-Cap Universe.

ANALYST DISCLOSURES

I, David Bautz, PhD, hereby certify that the view expressed in this research report accurately reflect my personal views about the subject securities and issuers. I also certify that no part of my compensation was, is, or will be, directly or indirectly, related to the recommendations or views expressed in this research report. I believe the information used for the creation of this report has been obtained from sources I considered to be reliable, but I can neither guarantee nor represent the completeness or accuracy of the information herewith. Such information and the opinions expressed are subject to change without notice.

INVESTMENT BANKING AND FEES FOR SERVICES

Zacks SCR does not provide investment banking services nor has it received compensation for investment banking services from the issuers of the securities covered in this report or article.
Zacks SCR has received compensation from the issuer directly, from an investment manager, or from an investor relations consulting firm engaged by the issuer for providing non-investment banking services to this issuer and expects to receive additional compensation for such non-investment banking services provided to this issuer. The non-investment banking services provided to the issuer includes the preparation of this report, investor relations services, investment software, financial database analysis, organization of non-deal road shows, and attendance fees for conferences sponsored or co-sponsored by Zacks SCR. The fees for these services vary on a per-client basis and are subject to the number and types of services contracted. Fees typically range between ten thousand and fifty thousand dollars per annum. Details of fees paid by this issuer are available upon request.

POLICY DISCLOSURES

This report provides an objective valuation of the issuer today and expected valuations of the issuer at various future dates based on applying standard investment valuation methodologies to the revenue and EPS forecasts made by the SCR Analyst of the issuer's business.
SCR Analysts are restricted from holding or trading securities in the issuers that they cover. ZIR and Zacks SCR do not make a market in any security followed by SCR nor do they act as dealers in these securities. Each Zacks SCR Analyst has full discretion over the valuation of the issuer included in this report based on his or her own due diligence. SCR Analysts are paid based on the number of companies they cover. SCR Analyst compensation is not, was not, nor will be, directly or indirectly, related to the specific valuations or views expressed in any report or article.

ADDITIONAL INFORMATION

Additional information is available upon request. Zacks SCR reports and articles are based on data obtained from sources that it believes to be reliable, but are not guaranteed to be accurate nor do they purport to be complete. Because of individual financial or investment objectives and/or financial circumstances, this report or article should not be construed as advice designed to meet the particular investment needs of any investor. Investing involves risk. Any opinions expressed by Zacks SCR Analysts are subject to change without notice. Reports or articles or tweets are not to be construed as an offer or solicitation of an offer to buy or sell the securities herein mentioned.

CANADIAN COVERAGE

This research report is a product of Zacks SCR and prepared by a research analyst who is employed by or is a consultant to Zacks SCR. The research analyst preparing the research report is resident outside of Canada, and is not an associated person of any Canadian registered adviser and/or dealer. Therefore, the analyst is not subject to supervision by a Canadian registered adviser and/or dealer, and is not required to satisfy the regulatory licensing requirements of any Canadian provincial securities regulators, the Investment Industry Regulatory Organization of Canada and is not required to otherwise comply with Canadian rules or regulations.